the other coats of these canals. The symptoms are fully explained by this hypothesis. In this connection it is well to call attention to a recent able work by Heubner (*Die Leutische Erkrankung der Hirnarterien*), in which a very clear account is given of the clinical history and pathology of syphilitic lesions of the vessels of the brain. A study of this work will materially assist the observer in the correct understanding of the symptoms of syphilitic cerebral disease, which might otherwise appear obscure.

125 EAST TWELFTH ST., November, 1875.

ART. III.—THE PATHOLOGY OF TETANUS.

BY H. M. BANNISTER, M. D., CHICAGO.

TETANUS is a disease with which, fortunately, the majority of the profession have not an extensive practical acquaintance. Its fatality and the mysterious character of its phenomena have, nevertheless, strongly directed the attention of medical men to its study and observation. The result of all this has been to clear up many points in regard to it, and, with the advances in therapeutics, has made its prognosis, in many respects, more favorable; but at the present time the disorder is yet much involved in obscurity, and the most varying views are still held in regard to its true pathology.

The prevalent idea in the medical profession at the present time may, perhaps, be stated as follows: Tetanus is an irritative functional affection of the spinal cord, extremely acute in its nature, and causing severe, long-continued and painful contractions throughout the muscular system. Beyond this most persons, I think, would feel it safest not to go in their definition of the disease.

It is the object of the present paper to offer, and support by facts and authorities, the theory of the pathology of tetanus, which, though not new, and perhaps not absolutely demonstrable, seems yet to meet the conditions of a satisfactory explanation as well as any that has so far been proposed. It is impossible, of course, to anatomically prove the initial nervous lesion in many cases of tetanus, and to follow it through the nerve trunks to the nervous centres from which the first manifestations of serious trouble generally appear. The only thing that can be done is to take the facts that are known, and find a hypothesis which will satisfactorily account for them, both singly and in their connection with each other, in the general *symptom-complex* of the disease.

As a rather typical case showing many of the usual features of the disease in its acute form, and also serving rather better than many others to illustrate what seems to me to be its true pathology, the following observation is offered. I am indebted for the greater part of the history to the courtesy of the attending physician, Dr. N. S. Davis, who kindly placed his notes at my disposal, and to that of Dr. J. S. Jewell, who was called in consultation during the progress of the case.

Mr. F. M. B., a theological student, well built and strong, and always previously healthy, aged about 24, stepped on a rusty nail in the early part of May, 1875, about the 11th or 12th of the month. The nail passed through the thin sole of his shoe, causing a penetrating wound on the anterior part of the bottom of his left foot, giving him some pain and inconvenience, but not enough to prevent him from going about and attending to his ordinary occupations. About the 15th or 17th of the month he went some distance in the country, and on his return his wound became more troublesome, and he was obliged, part of the time, to wear a slipper instead of a shoe, and to use a cane in walking. He felt what he took to be rheumatic pains passing up the leg and side of the body, appearing also under the scapula of the left side.

About the 19th or 20th of May he began to feel some stiffness of the lower jaw and the side of the neck, affecting, it was said, only one side at first, drawing his jaw slightly to that side. I have been unable to obtain from his friends any positive information as to which side was first affected; the impression, however, was, that it was the right side. It was not very prominent, however, and the trismus very soon became symmetrical. During this time he was still walking about, and had consulted a leading homœopathist, who attributed his troubles to inflammatory rheumatism, and continued to treat him until the afternoon of the 23d of the month, when the symptoms having become more serious, and the patient and his friends both recognizing the serious nature of the disorder, he was dismissed, and Dr. N. S. Davis was called in to take charge of the case. The condition of the patient, when first seen by Dr. Davis, was as follows:

He was lying in bed, in the dorsal position, the head drawn backward from the rigidity of the nuchal muscles, jaws firmly clenched and admitting of, at the best, only a separation of about the eighth of an inch; the face flushed, skin moist, and its temperature only moderately elevated; pulse 110 per minute, and moderately full; respiration natural in frequency, but more abdominal than usual. The patient complained of a sense of tightness across the chest, and of inability to move the head either forward or laterally, and of paroxysms of pain shooting from the epigastrium to the upper dorsal region of the spine, and also of some degree of headache. His mind was clear and his spirits good. The following prescription was ordered:

℞ Bromide of Potassium	ʒ vi
Hydrate of Chloral	ʒ vi
Tinct. Calabar Bean	℥ i
Syr. Wild Cherry Bark	℥ ii
Water	℥ iii

Mix. A large-sized teaspoonful to be given every half hour until there is either relaxation of the muscular rigidity or sleep. If either of these effects is induced, the interval between the doses to be lengthened to one hour.

May 24th, morning.—The patient slept some during the night, and the muscular rigidity was so much less that he could separate his teeth a quarter of an inch and move his head a little from side to side, but not forward. In all other respects his condition was the same as on the previous afternoon. The urine was scanty and high-colored. He was directed to continue to take the prescription before given, every two hours, and a teaspoonful of the following every intermediate hour:

℞ Tinct. Digitalis	℥ i
Nitrous Ether	℥ ii
Tinct. Aconit. Root	ʒ iss
Syr. Wild Cherry Bark	℥ i

Mix.

On the evening of the same day the symptoms were still farther improved in a moderate degree, and the same treatment was continued.

May 25th, morning.—The patient slept at short intervals through much of the night, and the muscular rigidity was so much relaxed that he was able to separate the jaws a full inch, and take nourishment and drink with facility. The head could be moved from side to side and slightly also forward. He complained of no pain, except occasionally in the cervical and dorsal regions of the spine; the urine was more abundant, but the bowels were still obstinately constipated. Directions were given to continue the administration of the chloral and calabar bean mixture every two hours, and the digitalis and aconite every four hours.

At the evening visit the patient still appeared comfortable, and no change was made in the treatment, except to give one powder containing five grains of calomel and eight of Dover's powder at bed-time.

May 26th, morning.—During the first half of the night, the patient rested well, and in consequence his medicine had been given at longer intervals. About 1 o'clock in the morning, however, he had a renewal of tension and distress from the epigastrium to the spine, with increased opisthotonos. The paroxysm was quite distressing, but lasted only a few minutes, and was followed by slighter paroxysms every half hour or hour. The pulse was smaller, and 120 per minute. In the interval between the paroxysms the muscles of the neck and jaws became considerably relaxed.

These symptoms appearing less favorable, Dr. J. S. Jewell was called to see the patient in consultation. He advised a faithful continuance of the chloral and calabar bean prescription, counter-irritation over the upper part of the spine, and enemas to move the bowels. Careful attention also to be given to nourishment, as on the preceding days.

At the evening visit the patient was found to have passed the day more comfortably, with less frequent paroxysms, but his pulse remained quick and weak; and it was reported, for the first time in the case, that he had two or three times exhibited slight mental wandering. The enemas had produced but one moderate movement of the bowels. Both urine and fæces were nearly natural. During the last forty-eight hours it appeared that an increase of fever, heat and general aggravation of the symptoms had occurred about 3 P. M. and 1 A. M. each day. It was therefore decided to add to the previous treatment, five grains of sulphate of quinine every six hours.

May 27th, morning.—Dr. Jewell was again called in consultation. As usual, the patient had been quite comfortable, and had had short intervals of sleep during the first half of the night; but from 1 to 3 in the morning, he had several paroxysms of renewed opisthotonos with rigidity of the diaphragm, accompanied by a sense of exhaustion. At 6 A. M., the time of the visit, there was only moderate rigidity of the cervical and dorsal muscles; the skin was moist; the temperature nearly natural; the pulse 115 per minute, and soft; respiration soft; the mind for the most part clear, but with occasional slight wanderings; urine scanty and voided with some difficulty; bowels quiet. It was decided to make no material change in the treatment except to induce a further movement of the bowels by enema during the morning, and as it was necessary for Dr. Davis to be absent from the town during the day, Dr. Jewell was to call and see the patient again at 2 P. M.

At two o'clock Dr. Jewell found the patient in nearly the same degree, as regards muscular rigidity, as in the morning, but presenting a decided increase of febrile heat and restlessness. The bowels had moved freely after the enema in the morning, and he had been subjected to an injudicious amount of moving and changing of position, in consequence of which he had had several severe paroxysms of spasmodic action of the dorsal muscles and diaphragm. On account of these symptoms of increased heat and spasmodic action, Dr. Jewell applied ice-bags to the spine and omitted the chloral and calabar bean mixture for four or five hours.

At first the application of ice to the whole length of the spine appeared to make a favorable impression, lessening the fever and pain and feeling comfortable to the patient. In about two hours, however, he began to complain of a sense of discomfort from their use, and at his request the ice-bags were removed. At 7 P. M. the patient was again visited by Dr. Davis and found to be inclined to be drowsy, with only moderate continuous rigidity of the muscles of the neck and back, but this was subject to slight spasmodic increase about every half hour, and greatly aggravated by attempts to change position. The pulse was 120 per minute, rather feeble; the bowels had moved three times; the contents were very liquid, and the last evacuation was partially involuntary. The pulse was also noticed to be unsteady and the mind more wandering. An anodyne and cordial mixture was immediately directed, with the hope of checking the looseness of the bowels and sustaining the strength of the patient. About an hour later the patient was again visited in company with Dr. Jewell. He was then quiet with but little continuous muscular rigidity, but inclined to be drowsy and mentally wandering, and with a very feeble and variable pulse. He had had one or two more thin and partially involuntary discharges from the bowels. The failure of the patient was evidently accelerated by the copious intestinal discharges. While, however, a prescription was being written, and the patient was in the act of taking a spoonful of drink, he was seized with a violent tetanic spasm, causing complete rigidity from head to foot. Both Drs. Jewell and Davis took hold of his pulse, which was for a moment as full and perceptible as before, or more so; then ceased instantaneously after one or two slightly irregular beats; the respiration, however, continuing for at least a minute or a minute and a half after the complete cessation of the heart's action. Dr. Jewell carefully tested the question as to whether the cardiac action had ceased or not by direct auscultation, but no pulsations could be detected after the cessation of the radial pulse. The pupils were somewhat dilated, but no change in them could be detected at the time.

Owing to the objections of the friends, only the most hurried and superficial post-mortem examination could be made. Still it was thought best to utilize the opportunity as far as it was allowed, and it was made the next morning, some thirteen hours after death, by Dr. Jewell, assisted by the writer. The following were the principal points noted:

Post-mortem rigidity well marked. Features natural, no traces of the disease appearing as far as they were concerned. All dependent parts, the back, the nates, and the posterior aspect of the limbs much discolored by suggilation. The wound on the foot seemed to have almost entirely healed; the scar was not at all noticeable.

On opening the cranium the dura mater appeared of a dark leaden color, thickly specked with minute drops of blood oozing through its substance and tense from the contained fluid. When it was opened a large amount of dark bloody serum or very liquid blood, nearly a pint or more, escaped. The spinal canal was not opened, but the cord was cut as low as possible through the foramen magnum. It seemed somewhat softened in its cervical portion as far as could be seen; the membranes appeared inflamed, and a purulent appearing fluid was continually trickling out of the spinal canal. No microscopic examination was made.

Reviewing the order of phenomena in the above case, we find first, a peripheral wound of the foot irritated by constant use of the member, and producing, in all probability, a local neuritis of the sensory fibres of the foot and leg; next, without any intermediate symptoms, excepting some rather indefinite pains in the shoulder, as far as could be learned, tonic spasm of the muscles of mastication, at first unilateral, then, becoming bilateral, the contraction involving successively the muscles of the neck, the back and the diaphragm. After a day or two of treatment these symptoms gradually became much improved, the jaws relaxed, and the head became more movable, the pain and discomfort largely disappeared. The extremities were all this time uninvolved. Then, without any very apparent cause, we have an aggravation of the symptoms not attended with any special increase in the permanent contraction, spasms of opisthotonos and general muscular rigidity, accompanied with painful sensations of diaphragmatic tension, great increase of temperature, and, what is a peculiar feature in this case, slight cerebral symptoms. The patient finally succumbed in one of the attacks, apparently from paralysis of the heart, in the general spasm. During this time the primary peripheral injury was apparently healing, for while the foot was still painful and swollen when the rigidity of the jaws first appeared, it seemed perfectly normal, without any very marked cicatrix at the time of death. The closing diarrhœa may be considered, I think, as an incidental phenomenon, not so directly connected with the central disorder as were the other symptoms. Though it had its part in reducing the patient's strength, and thus preparing for a fatal termination, this actually occurred from an apparent accident entirely independent of it, but one which we think is often a cause of death in this disorder.

The case is sufficiently typical to answer as an example of the general course of tetanus, and yet it presents peculiarities by reason of which it is, in my opinion, more suggestive as to the pathology than many others. Taking up the phenomena in the order in which they made their appearance, we have, first, what probably always occurs in traumatic cases, though not always recorded, a local irritative affection involving the sensory nerve fibres—a local neuritis, in short. This, in the present instance, may have been entirely transient in its nature; certainly no symptoms of its existence were observed after the central disease had become pronounced. That a neuritis, or at least a local irritation of sensory nerves, is often the original lesion in tetanus, we have ample evidence, and strong inferential proof that this is always the case. Thus, besides the symptoms of irritation, such as were observed in this case, we have as evidence the actual results of examinations, such as those reported by Curling,[*] Lepelletier,[†] Froriep,[‡] Demme,[§] Michaud,[¶] Remak,[‖] and others.

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[*] *A Treatise on Tetanus*, London, 1836. Abstr. in *Schmidt's Jahrb.*, XVIII., 1838, p 264.

[†] *Revue Medicale.* IV., 1827, } Quoted by Friedreich, *Ueber prog. Muskel-*
[‡] *Neue Notizen*, 1837. } *atrophie.*

[§] *Beitraege zur path. Anat. des Tetanus*, Leipzig, 1859. Quoted by Thamhayn, *Beitr. zur Lehre von Tetanus. Schmidt's Jahrb.*, 112, 1861, 210.

[¶] *Archives de Physiol. Norm. et Path.*, No. 1, 1872.

[‖] *Med. Central-Zeitung*, XXIX., 12, 1861, p. 210.

One of these observers, Demme, traced, in one case, the lesion from the point of injury to the cord, along the course of the nerve; it being in this instance a kind of chronic neuritis characterized by proliferation of the connective tissue. Then we have the good results obtained in some cases by section of the nerve above the point of injury, thus cutting off the morbid process between the periphery and the nervous centres. That this is only an exceptional result does not diminish the value of the fact as a proof of the nervous irritation; it is rarely practiced early enough in the disease to justify the expectation of its being efficacious in cutting it short. Nor is it needful to suppose that the neuritis is continuous from the periphery to the centres; it is in all probability very rarely the case that it is so, and I have elsewhere* endeavored to point out that irritative action readily passing into inflammation, under favorable circumstances, may traverse nerve trunks and leave no permanent trace. That such is the case in tetanus, I have no doubt.

It is necessary to suppose, however, that the irritation in tetanus is of a peculiar character in some respects. There is reason to think that it follows exclusively the sensitive fibres and very probably those alone which have direct connections with the peripheral point of injury. The great majority of observers have classed the phenomena of tetanus among the reflexes—it is hardly necessary to quote authorities in regard to this point. The fact that wounds in localities rich in sensory nerves are those which are especially liable to be followed by tetanus, while in others less richly provided they are much less dangerous in this respect, is significant. Thus, the extremities, the face, and the genitals are the regions in which wounds are most likely to be followed by this disorder, and each of these is specially supplied with sensory nerves. It is not at all necessary that there should be any consciousness of this irritation other than that of the original wound, to which necessarily all sensations carried to the centres along these particular fibres would be referred.

The routes of sensory transmission in the cord, as shown by the experiments of Schiff, Brown-Sequard and others, lie chiefly in the central gray matter in which they decussate at different heights, and this brings us to the second point in the history of our case, the unilateral trismus. Tetanus is, as I have stated in the beginning of this paper, usually regarded as a spinal disease. This idea is strengthened by the constant comparison of the toxic spinal action of certain poisons, such as strychnine, etc. Some very high authorities, such for example as Leyden,* have declared that tetanus is a functional disease of the cord altogether analogous to the toxic tetanus of strychnine without any constant perceptible alterations to be found after death. Curling also defined the disease as a functional affection of the motor tracts of the cord without any constant lesion. It would seem that attention to the fact, that in nearly all cases the first manifestations of rigidity appear in a region not innervated by spinal nerves, should have corrected this view. The vulgar name of the disease—lock-jaw—indicates this fact. In the case reported, apart from the evidences of irritation of the peripheral nerves, this was the first phenomenon, with the added peculiarity that in the beginning it was said to be unilateral, the jaw being drawn to one side, that opposite the original lesion in the foot, though the contraction very soon became bilateral. This is a peculiarity which is not mentioned by many authorities, but it is only what might be expected to appear in some instances of this disorder, if we are to suppose that the irritation travels to the centres along the sensory fibres. In this case the onset of the tetanic symptoms was more gradual than in most cases, and this special feature is, I think, due to that fact.

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* This journal, Oct., 1875.

The explanation of this almost invariable primary involvement of the masticatory muscles must be, that the irritation finds its first point of attack on the great nerve centres somewhere in the region of the medulla or pons. It cannot, therefore, be called, exclusively at least, a spinal disease; that the spinal symptoms are most prominent is simply due to the fact that it affects the muscular system generally, and that is nearly everywhere directly innervated by spinal nerves. In fact, it is as easy to believe that the spinal symptoms may be only secondary as it is to consider them as constituting the real trouble in tetanus. And here comes in the distinction between the toxic tetanus of strychnine and the pathological condition under consideration. It has often been demonstrated that the tetanic convulsions of strychnia poisoning are spinal, and not cerebral, and that section of the cord has no effect upon them except perhaps to render them more intense in the points thus removed from cerebral influence. Moreover, in the toxic tetanus the order is reversed: the convulsions appear at the first and the jaws only become fixed at a very late stage, and after the action of the drug has long been marked on the muscles of the trunk and extremities. These differences seem to have been overlooked to a very great extent by those who make the comparison between the tetanus from strychnia and that from traumatisms, or the so-called idiopathic form.

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* *Virchow's Archiv*, XXVI., 1863, 538.

That the original seat of the disease is in the medulla or in the basal ganglia of the brain, has been recognized by many authors. Among others I may mention Neumann,* Warnecke,† who considers that it may be in the anterior roots of the medulla, and Luys‡ who locates the first central disturbance in what he calls the convulsivant region *par excellence*, where the fibrous networks from the gelatinous substance of the floor of the 4th ventricle and the cerebellar peduncles are most multiplied, and where, consequently, the tension of cerebellar and excito-motor innervation should be the greatest. This of course involves the theory of Luys as to the functions of the cerebellum—that it is a reinforcing ganglion—which may not be accepted by the reader; I have quoted it only as giving his opinion as to the original cerebral seat of the disorder. Demme§ also maintains the theory that the primary origin of tetanic symptoms is to be sought for in the medulla, which he holds must be in a predisposed irritable and sensitive condition, and lastly Poincarè,‖ in his recent admirable work on the nervous system, locates its seat in the pons. These references might be multiplied, but they are sufficient of themselves to show that the objections to the exclusively spinal theory of tetanus have not been universally overlooked. It is a matter of surprise, however, that so many writers who have studied the subject, and who adopt a neurogenic theory of the disease, have, at least in their language, left it to be supposed that it is mainly and exclusively a spinal malady.

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* *Krankh. des Menschen*, Berlin, 1838. *Schmidt*, XXXII., 1841, 259.

† *De Tetani Causa et Natura*, Gottingen, 1840. *Schmidt*, XXX., 1841, 238.

‡ *Recherche sur la Syst. Nerveux*, Paris, 1865, 599.

§ *Militar. Chirurg. Studien*, Wurzburg, 1861. *Schmidt*, CXI., 134.

‖ *Lecons sur la Physiologie, Normale et Pathologique du Systeme Nerveux*, II., Paris, 1874, pp. 104 et. seq.

The mechanism of the transmission of the irritation from the peripheral wound to the centre for the motor root of the fifth nerve, which is specially concerned in the production of this early symptom of trismus, cannot be stated with certainty. There is no reason however, to suppose that nerve fibres can convey irritations differing otherwise than in degree, at least none that rests on any certain knowledge, and inference is strongly the other way. The terminal apparatus is probably what makes the difference in the effects. Nor is it possible to say why the irritation in this case affects especially the nucleus of the motor division of the trigeminus; we are left very much to conjecture as to this point. We do know, however, that in the floor of the fourth ventricle and in the gray matter in which this nucleus is situated, is a centre from which may be excited general convulsions of the whole body, and though this fact does not afford an explanation of a tonic spasm of a special set of muscles, it is nevertheless full of suggestiveness. Nothnagel* has, by careful experiments, not only fixed the general limits of this region, but shown that in reality it is only a centre from which reflex convulsions can be the more readily excited than elsewhere, and that the real centre must be sought higher up, probably in the substance of the pons. These facts certainly show that the region of the medulla and pons is specially excitable, and will account in some degree for the earlier participation of the facial muscles than those of other parts of the body.

There is, however, another point that is worthy of consideration here. The rigid contractures that are often met with in various kinds of central nervous disorder are, I think, in a large majority of cases the result of cerebral and not of exclusively spinal disease,—especially is this true of such as are of rapid development, like those of tetanus. The cord may be

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***Die Enstehung allg. Convulsionen, etc.* *Virchow's Archiv*, XLVI., 1868, p. 1.

and, perhaps, generally is, affected, but as a secondary result of the brain trouble. It certainly is functionally affected. Thus we have the sudden contractures observed to follow cerebral hæmorrhages, which may follow lesions of the pons, the medulla, and the crura, as well as those of the corpus striatum, and many forms of the so-called hysteric contractures, with catalepsy and similar phenomena. Then it is worthy of remembrance, also, in this connection that, according to the system of Meynert, the chief reflex centres for the body are located in the thalami and corpora quadrigemina, the fibres conducting the reflex influences passing downward to the medulla in the tegmentum or upper portion of the crus. Lastly, we may mention the recent experiments on the motor functions of the cortex, which may *possibly* have something to do in some cases with the phenomena of tetanus and allied diseases.

The whole subject of the more remote pathology of contractures is involved in so much mystery, that at present it is impossible to give any very satisfactory theory that rests upon actual known facts in regard to it. It is possible that it depends upon very different conditions in the brain and spinal cord at different times. In the majority of cases of tetanus, however, there is no doubt that it begins from a cerebral cause.

The reported unilateral character of the tonic spasm in the case I have related justifies the supposition that the irritation that directly caused it may have been higher in the nervous centres than the medulla. Its rapid change to a symmetrical trismus, and the fact that it is so as a general rule in such cases, point as strongly that the medulla or, at least, the pons is the seat of the principal reflex trouble. The stiffness of the muscles of the neck, which appears generally almost simultaneously with the masticatory spasm, also depends, in part at least, on irritation of a nerve with intracranial connections, the spinal accessory nerve of Willis. The rapidly following stiffness in muscles of the trunk may be, and probably is due to a gradually descending progress of the morbid process in the cord. Why, with this general trouble in the cord, the arms and hands should still preserve their mobility, and generally, as in this case, the legs, also, is not very easily accounted for. In other cerebral and spinal diseases, both contractures and paralysis are more apt to be noticed in the members than in the trunk.

In our case the improvement in the symptoms of this stage of the disease is rather notable. The jaws relaxed, the head became movable, and the patient was able to pass part, at least, of the night in refreshing sleep, and to take nourishment, the epigastric distress disappeared or greatly abated, and the condition was generally more comfortable than is often the case in this disease. This improvement continued until the sudden outbreak of the third or convulsive stage on the morning of the twenty-sixth, which was ushered in by a renewal of the epigastric distress and a paroxysm of increased opisthotonos, which latter was repeated every half hour or hour thereafter, the muscular rigidity continuing somewhat relaxed in the intervals. In these respects, especially in the mildness of the second stage, the case is peculiar; tetanus often, indeed, proves fatal or recovers without reaching the stage of convulsions at all.

It is difficult to explain the sudden change in the symptoms from a continuous, though gradually lessening rigidity, to rapidly recurring convulsive seizures, without inferring some serious structural lesion, equally sudden in its full development. And here the convulsive centre in the medulla may, perhaps, be called in to interpret the symptoms, or rather, perhaps, a general lesion affecting the excitability of the reflex centres throughout the cord, rendering them more readily responsive to impulses from the general reflex centres in the medulla or basal ganglia of the brain. I am inclined myself to refer either the beginning or the full development of the myelitis, etc., revealed in the autopsy, to this period, as the change from an irritative functional disorder to an actual structural lesion. The cerebral symptoms, also, which began to be manifested at this time are explained by the participation of the brain in the general highly congestive condition.

The increase of heat noted at this stage of the disease is what is often observed in tetanus, and has been the subject of very numerous and extensive investigations. The best discussion and *resumé* of this subject is that given by Rose,* who, after noticing the experiments and statements of Leyden † and others who have sought to show that this increase of temperature was due to muscular contraction, he offers the conclusion that it is not a direct consequence of the muscular affection, but that it indicates the central locality of the whole disorder—that it is due to the exhaustion of the nervous centres, and especially of the heat regulating centre in the medulla or pons. It is, therefore, an additional testimony in favor of the theory of the central seat of this disease, which I have endeavored to demonstrate. The facts brought forward by Rose that many cases of tetanus pass to a fatal termination without this increase, and that it appears with the convulsions in the latter stages of the affection, supports this view. Verneuil‡ has likewise pointed out that a sudden rise of temperature is a very grave prognostic in this disease, and the explanation by this theory of the exhaustion of the higher nerve centre is not altogether inconsistent with the one he gives that death is due to acute phlegmasic pulmonary alterations due to the reflex action of the cord on the pulmonary and bronchial vaso-motors. If the view expressed by Wood,§ that this superior regulatory heat centre acts on subordinate ones in the cord as a controlling or inhibitory power, be true, we then have a still more obvious explanation of the rise of temperature in an irritative affection like tetanus, involving the whole cord in its progress.

Lastly in our review of the case, we have the mode of death, which was not the usual one. The most frequent cause of death in tetanus, according to Rose, is exhaustion, next is asphyxia from fixation of the respiratory muscles, and these two causes are all that are recognized by many writers on this disease. Neither of these was the cause of death in this case. The fatal termination occurred very suddenly, without any premonition, from arrest of the heart, respiration continuing at least eighty or ninety seconds longer. Rose admits paralysis of the heart as a cause of death in this affection, but he attributes this cardiac stoppage to the resistance of the generally contracted muscular system to the circulation during the convulsion, but not to any participation of the heart in the same. As evidence of this, he says that at the conclusion of the spasm the pulse momentarily returns to its normal condition. Nothing of the kind occurred in the case I have given; the pulse from being, perhaps, even a little fuller and stronger than before, gave one or two irregular, convulsive beats, and then stopped entirely and permanently, not being detected either by palpation or auscultation during the short continuance of the convulsion, or in the general muscular relaxation which followed it.*

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* Pitha-Billroth, *Chirurgie.* Erster bd. zweite abth , I., 3, pp. 21–39.

† *Zur Path. des Tetanus*, *Virchow's Arch.*, XXVI., 1863. 538.

‡ *Soc. de Chirurgie*, *Seance* of Oct. 30, 1872. *Bull. generale de Therapeutique*, LXXXIV., 1873. P. 41.

§ *Toner Lectures*, No. 4. Jan., 1875.

Other authors, such older ones as Travers, Currie and Howship,† and more recently Thamhayn,‡ have admitted the occasional occurrence of tetanus of the heart, and cases have been reported in which the heart has been found closely contracted shortly after death. There seem to me to be no very good *a priori* reasons for absolutely excluding the possibility of tetanic spasm of the heart, though it would seem that it *may* have to depend upon the intrinsic nervous apparatus of the organ, and not on the spinal cord. Though we know certainly, perhaps, of no direct spinal excitation that can arrest the heart in systole, this may not be necessary to cause an arrest of the heart in tetanus, since the morbid action working on the nucleus of the pneumogastric may perhaps produce its stoppage in diastole, without calling in any such mechanical agency as the resistance of the external circulation, which, it seems to me, would in many cases produce actual rupture. Nor is it very probable that such a density and contraction of the cardiac tissue as has been reported as having been observed after death, could have been due to mere post-mortem rigidity. In the case related I am inclined to consider death as due to direct central interference with the cardiac innervation, but whether by paralysis by inhibition, or stimulation into spasm, cannot be said with certainty, though I am inclined to believe the first rather than the second.

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* Since writing the above paragraph I have seen the memoir of Bauer, contained in the twelfth volume of *Ziemssen's Handbuch der spec. Pathologie und Therapie.* This author states that stoppage of the heart is the cause of death in the majority of cases, and that the danger from embarrassment of the respiration is generally overrated. In this he differs from most authors, but is, perhaps, correct. The actual observation of the cardiac arrest is certainly not recorded in most reports of cases of this disorder.

In this connection, I must also acknowledge my indebtedness to Dr. J. S. Jewell, for the use of his very extensive library, in which I obtained access to this and many other works, in the preparation of this paper.

†Quoted by Romberg and Watson.

‡*Schmidt's Jahrb.*, CXII., 1861, 210.

The appearances observed at the autopsy in this case, as far as they go, are of importance. They amount simply to this: acute congestion of the brain and inflammation and disorganization of the cord. The first of these will account for the slight cerebral symptoms which, when present in tetanus, are generally attributed to defective action of the lungs or to the remedies employed. Neither of these had probably any agency in their production here.

The appearances on the part of the spinal cord, though very imperfectly observed, being confined to the microscopic examination alone, denoted very serious inflammatory trouble, occupying at least the cervical region. The lack of a microscopic examination prevents me from saying any more in regard to it than that it was a very positive and satisfactory evidence of a serious structural lesion of the spinal cord and its membranes. Post-mortem examinations in this disease have been so various, and in many cases so unsatisfactory in their results, that this case is almost a brilliant exception, even with the very hasty and imperfect examination that was made.

The fact seems to be that the structural lesion in tetanus may range from absolutely nothing that can be detected after death, to the most complete inflammation and disintegration of the cord, with sometimes extravasations in the substance of the nervous centres and increase of their connective tissue, and inflammation of their enveloping membranes. Rokitansky* and Demme† have sought to find the anatomical lesion of tetanus in a sclerosis. Lockhart Clarke* has found what he considered a constant lesion in local granular disintegrations of the cord; and Michaud† makes its characteristic lesion in a subacute myelitis of the central gray matter of the cord. All these observers have specially investigated the spinal cord, any such extensive alterations of which, according to the views I have expressed as to the original seat of tetanus, must be the secondary and not the primary lesion. Looking at the subject in the light of the physiological functions of the parts involved, it would seem that the seat of the spinal structural lesion should be that described by Michaud, since in the central gray matter are contained the parts principally involved, viz.: the spinal reflex centres and the routes of sensory transmission. But the brain has not been found always normal; the principal lesion described is congestion in spots or patches, which have been found by Liouville‡ in the gray matter of the thalami, the corpora striata and even in the convolutions. That in many cases no change at all has been detected, may perhaps be due to imperfections or a want of thoroughness in the examinations; or it may even be that whatever lesion exists may be in some cases of such a character as to be absolutely undetectable with our present means of observation. We cannot, nevertheless, avoid admitting at least some change to correspond with the serious functional disturbance, at least in such cases of tetanus as run an acute and fatal course.

**Sitzungsber. des K. K. Akad. der Wissensch.*, Wien, 1857, 1 & 2. 517-536.

†*Beitraege zur path. Anat. des Tetanus.* Bern, 1857. Both quoted by Leyden.

Summing up now the principal points of the case given and their discussion in the preceding pages, it appears that the course of the great majority of cases of tetanus may be stated as follows:

1. A peripheral wound involving the sensitive nervous fibres and causing an irritative local lesion—a neuritis.

2. Transmission of this irritation, either merely as such, or as actual inflammation, through the nerve trunk and the gray matter of the cord to the medulla and pons, and possibly to the higher reflex centres in the optic thalamus and corpora

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***Med. Chir. Trans.*, XLVIII., 263.

†*Archives de Phys. Norm. et Path.*, 1872, No. 1.

‡Quoted by Vulpian. *Leçons sur l'Appareil Vaso-Moteurs*, II., 1875, p. 93.

quadrigemina, or perhaps even to the cortical motor centres of the brain.

3. Reflection of this irritation along motor nerves, at first only the trigeminus and accessory, then gradually involving other and spinal nerves and producing tonic contraction of nearly all the muscles of the trunk. This is the condition till death, in many cases of the disease, but in others convulsions appear in a late stage of the disease.

Death may occur from exhaustion, asphyxia, or paralysis of the heart. This latter is probably due to involvement of the cardiac innervation in the disease, and to direct implication of the nucleus of the vagus nerve, although it may possibly be due to a cardiac tetanus.

These, stated in the briefest and baldest manner, seem to me to contain the principal points of the pathology of tetanus. In many respects a greater accuracy and refinement in their statement is impossible ; we do not know exactly the kind of irritation transmitted nor the exact mechanism of the central apparatus set in action by it. I will only say, in addition to what has been said in the discussion of the case, in regard to the part of the medulla in the production of the tetanic accidents, that the vaso-motor centres, and the principal one in the floor of the fourth ventricle especially, may in some cases have something to do in bringing about these results. This idea has been advanced by Benedikt* and is thought not impossible by Bauer in his recent excellent memoir on tetanus in the twelfth volume of Ziemssen's *Handbuch der spec. Pathologie u. Therapie*, which has been received since the first part of the present article was written. As I have already said, the above is the probable pathology of the great majority of cases of tetanus, *i. e.*, those depending on a peripheral lesion or irritation. And under this head I would include the greater number of the so-called idiopathic cases which depend none the less on an external irritation, for which an actual wound is not always needed. As neuritis may arise from a cold or a light contusion as well as from an actual traumatism, so tetanus, which follows a neuritis, in my opinion almost invariably, may have a similar origin.

**Nervenpathologie u. Elektrotherapie*, Wien, 1874.

There are some exceptions, however, to every rule, and the pathology of tetanus may not always be exactly as described. In some rare instances it may be of purely central origin; the morbid process may start in the cerebral reflex centres themselves. That these cases are rare the experience of the profession will testify, and when they do occur it is not often possible to identify them as such; we cannot exclude all possibility of an external irritation. But there is no reason why they should not occasionally happen, and I have no doubt that they are now and then met with. It may be that, as a general rule, the central irritations which might produce this trouble are either so serious as to cause a very sudden death, or so slight and transient as to produce only very temporary and insignificant symptoms. That this last is the case we have considerable evidence; the non-tetanic masticatory spasm of Romberg must be due to excitation of the same nerve regions as are concerned in the production of tetanic trismus; and this I think, in a slight degree, is not altogether rare from cerebral causes. A gentleman of my acquaintance has, on several occasions, had a slight tendency to fixation of the jaws, when suffering from fatigue caused by continuous mental exertion.

Again, there are occasionally instances in which the spinal reflex symptoms precede those which have their origin in the medulla. Such are the cases mentioned by Romberg, in which tetanic spasm is first noticed in the wounded member. In this case the independent reflex centres in the cord are first put in action, and the higher centres only secondarily. Still, even in these, the history of the case generally is that the characteristic trismus is observed before the general rigidity of the muscles of the trunk, thus showing that the implication of the cord, as a whole, is secondary to that of the medulla.

From what has been said it will be seen that the essential feature in the pathology of non-toxic tetanus is the lesion, either functional or structural, and probably both, of a region in the base of the brain which is concerned in the production of tonic spasm. If the view of Meynert be correct, this tract may be in the thalami or the corpora quadrigemina; but, at all events we must consider the region of the floor of the fourth ventricle, at least in a secondary way, as actively concerned in causing the symptoms observed in this disease.

There is one fact which bears against the theory of any necessary implication of the higher cerebral motor centres in this affection: that is its frequency in very young infants. It has been often stated, and the recent experiments of Soltmann* seem to confirm the statement, that in newborn infants existence is purely spinal and reflex. Now, though we cannot deny confidently with our present knowledge, a certain amount of reflex function to some portions of the cortical motor regions, and their participation in some cases of this disease, Soltmann has shown that these cortical motor centres do not functionally exist in newborn animals. The great frequency, also, of trismus neonatorum under favorable conditions for its occurrence, is also suggestive that the lack of the inhibitory action of the higher centres may have something to do in its production. The fact that contractures of the members from spinal disease indicate lesions in the lateral columns, which are the route of conduction of the motor orders of the will, likewise points this way.† I merely offer these as thoughts suggested by the consideration of the subject, not as probable facts.

The resemblance, in some particulars, between tetanus and hydrophobia, has led to a great amount of speculation and not a little erroneous conjecture. A number of authors, following out the apparent analogy between the two diseases, and the resemblance of the symptoms, in some respects, to those of poisoning by certain toxic substances, such as strychnine, caffeine, brucine, etc., have held that tetanus is a blood disease, and not an irritative nervous affection. They are at fault, in my opinion, first in attempting to use the more mysterious disorder to explain the one that is less so, and second, in ignoring certain points in which tetanus is different from all the troubles that have been compared to it. We have scarcely any evidence of the real lesion in hydrophobia, beyond the mere fact that its starting point is in a virus. Its symptoms in man have, as Bauer says, really little in common with those of the disorder in question, beyond the general reflex excitability and the pharyngeal spasm which, sometimes, though rarely, also occurs in tetanus. The distinction between the toxic effects of various drugs and the symptoms of tetanus, has been already pointed out, at least in part, in the preceding pages. And although a recent writer* has endeavored to show that some of these agents act upon the convulsive centre in the medulla, the distinction in the nature of the phenomena they produce is still apparent.

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* See Periscope of this number.

† The experiments of Woroschiloff (see Periscope), indicating the necessity of the integrity of a portion of the lateral columns for the production of tetanus are suggestive in this connection.

Among the authorities who have sought to show that tetanus is a blood disease, chiefly on account of its apparent resemblance to hydrophobia, I may mention Roser, Richardson, Spencer Wells and W. H. Thomson, none of whose articles I have seen, but who are all quoted by Thamhayn.† Other writers have considered it a zymosis on different grounds, chemical and otherwise. The peculiar endemic and epidemic relations of the disease on some occasions and in certain localities have also suggested this theory to certain writers. Some of the theories as to the pathology are worthy of mention only as among the curiosities of medical literature. Such for example is that of Hanke,‡ who considered it an affection of the central organs of the ganglionary system; that of Martin de Pedro,§ who called it a catarrhal rheumatismal disorder, and that of Forbes,‖ that it is "a result of an augmented disintegration of muscular tissue." These serve to show the range of ideas upon the subject, if nothing else.

I have nothing to say beyond what has been already said, in regard to the symptoms, the diagnosis and the treatment of tetanus; they are not the subject of the present paper. I have only endeavored to give what seemed to me the best explan-

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*Heubel, *Pflueger's Archiv*, IX., 6, *Das Krampfcentrum des Frosches u. sein Verhalten gegen gewisse Arzneistoffe*, pp. 263 et. seq.

†Loc. cit.

‡*Der Wundstarrkrampf*, anal. in *Schmidt*, XXV., 1840, p. 75.

§*Nueva Doctrina acerca del Tetanos y. de zu Curacion.* Madrid, 1869. Quoted by Poinçaré.

‖*Trans. of the College of Physicians and Surgeons of Philadelphia*, 1875.

ation of the pathology of the disorder, or at least the pathology of its more usual and typical forms. It will be seen that I do not consider it a purely reflex trouble, for that, in my opinion, would not necessarily require a structural lesion, which I believe to be always present in tetanus. It, nevertheless, in its manifestations and development, follows in a general way, the laws of reflex action. A complete understanding of this disease will perhaps, in time, lead to an elucidation of some still more obscure problems, such for example as the pathology of hydrophobia and hysteria.

Art. IV.—NOTES ON THE PATHOLOGY OF THE SYMPATHETIC NERVOUS SYSTEM.*

By T. Edwards Clark, M. D., New York City.

SOME months ago we began investigating the action of alchohol on the sympathetic nervous system, and in our preparatory work we were surprised to find how little is *definitely* known in regard to the pathology of this part of the animal structure.

We have been induced to present, in the form of the paper of the evening, a portion of our notes made while reviewing this subject.

Before entering upon the pathology of the sympathetic, we ought, perhaps, to allude briefly to its functions as ascertained by experimenting on the lower animals. In 1727, M. Pourfour du Petit found that after section of the cervical sympathetic, the pupil became contracted, the cornea flattened, the conjunctiva hyperæmic, the eyelids partially closed, and the eye appeared smaller and was drawn backward into the orbit. Similar experiments have since been made by Claude Bernard,

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* **Read before the N. Y. Neurological Society, Nov. 1st, 1875.**